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Convulsive Movements in Bilateral Paramedian Thalamic and Midbrain Infarction

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Key Words

Cerebral infarction · Convulsive movements · Seizures · Mesencephalic reticular formation

Abstract

Although some previous reports have described convulsive movements in bilateral paramedian thalamic and midbrain infarction, little is known about their nature. A 71-year-old man presented with impaired consciousness and clonic movements of both arms. Each series of movements lasted 10 to 20 s and occurred at 2- to 3-min intervals, which disappeared after intravenous administration of diazepam and phenytoin. Magnetic resonance imaging showed acute bilateral paramedian thalamic and midbrain infarction. A review of the literature revealed that convulsive movements were observed mostly at the onset of infarction. Clonic movements appeared frequently in the limbs, particularly in both arms. Clinical observations and results of animal experiments suggest that these seizures might originate from the mesencephalic reticular formation. Physicians should recognize this condition, because not only seizure control but also early management of ischemic stroke is required.

Introduction

Lesions in the thalamus and those extending into the midbrain can cause various movement disorders such as dystonia, asterixis, and ballism-chorea. Dysfunction of the neural networks involving the thalamus may contribute to these abnormal movements [1]. Convulsive movements have been reported in bilateral paramedian thalamic and midbrain infarction [2–7]. However, little is known about their nature. We here report on the case of a patient with clonic movements of both arms that were observed at the onset of infarction and offer a review of the literature.

Case Report

A 71-year-old man, with a ten-year history of chronic hemodialysis due to diabetic nephropathy, was brought to the emergency room. His wife found him unconscious beside his bed. On admission, his blood pressure was 185/86 mm Hg and his pulse was regular. He was in a coma but breathing spontaneously. His pupils were 4 mm in diameter and unreactive to light. There were neither voluntary eye movements nor eye movements in response to oculoccephalic reflex. There was no response to plantar stimulation. Spontaneous symmetrical clonic movements of both arms lasting for 10 to 20 s were observed at 2- to 3-min intervals. The abnormal movements disappeared after intravenous administration of diazepam and phenytoin. The results of laboratory tests on the following were normal: blood cell count, electrolyte level, glucose level, arterial blood gas, and cerebrospinal fluid. Electrocardiography results were normal. Brain magnetic resonance imaging (MRI) showed acute bilateral paramedian thalamic and midbrain infarction ([fig. 1](#)). MR angiography showed atherosclerotic changes in the internal carotid arteries and basilar artery. He was treated with argatroban. Electroencephalography (EEG) on the next day showed diffuse slowing. Ten days after admission, he opened his eyes following painful stimulation. He moved his limbs spontaneously and the horizontal oculoccephalic reflex was present. He was transferred to a rehabilitation hospital 17 days after admission.

Discussion

We report on the case of a patient with clonic movements of both arms at the onset of bilateral paramedian thalamic and midbrain infarction. In the literature [[2–7](#)], convulsive movements were observed mostly at the onset of infarction. Clonic movements appear frequently in the limbs, particularly in both arms ([table 1](#)).

Lesions in the thalamus and those extending into the midbrain can cause various movement disorders such as dystonia, asterixis, and ballism-chorea. The thalamus relays inputs from peripheral sensory pathways, the basal ganglia, and the cerebellum to the cerebral cortex. Lesions in specific thalamic nuclei and dysfunction of the networks may contribute to these abnormal movements [[1](#)]. However, dystonia, asterixis, and ballism-chorea are unlikely because of the clonic nature of the movements in our patient.

Although the cerebral cortex is considered important for the development of seizures in general [[8](#)], previous experimental studies have suggested that electrical or chemical stimulation of the brainstem tegmentum, particularly the mesencephalic reticular formation (RF), induces convulsive seizures [[9–12](#)]. Autopsy and MRI show the involvement of the midbrain tegmentum in patients with convulsive movements [[2–7](#)]. However, to the best of our knowledge, such movements have never been observed in bilateral paramedian thalamic infarction without midbrain lesions. These findings suggest that the mesencephalic RF has a crucial role in the generation of convulsive seizures in bilateral paramedian thalamic and midbrain infarction.

Convulsive seizures induced by electrical stimulation of the mesencephalic RF are not eliminated by precollicular transection [[10, 11](#)], but pontine tegmentum lesions attenuate the convulsions of brainstem origin [[13](#)]. These findings suggest that the generation of the motor component in convulsive seizures is induced by the activation of the descending RF. Furthermore, seizures can take a variety of motor patterns depending on the intensity of stimulation. A high intensity of stimulation causes tonic movements, whereas a low intensity of stimulation causes clonic movements of the forelimbs, and the subsequent increases in stimulation intensity produce a spread of

clonic movements down to include the hind limbs [14]. These motor patterns are also observed following spinal cord stimulation in decapitated animals [15]. It is hypothesized that the rostrocaudal spread observed in convulsions reflects the rostrocaudal spread of discharge down the polysynaptic descending pathways within the intermediate zone of the spinal gray matter [15]. This can explain why clonic movements were sometimes observed only in both arms, but have never been observed in the legs without the involvement of the arms in bilateral paramedian thalamic and midbrain infarction.

In summary, convulsive movements, particularly clonic movements of both arms, can develop in bilateral paramedian thalamic and midbrain infarction as an initial symptom. Physicians should recognize this condition, because not only seizure control but also early management of ischemic stroke is required.

Table 1. Convulsive movements in bilateral paramedian thalamic and midbrain infarction

Reference		Sex/age	Time	Movements	Method
Segarra [3]		M/51 years	Onset	Convulsive seizures	Autopsy
Castaigne [4]	Patient 3	M/51 years	Onset	Clonic movements of both arms	Autopsy
Ropper [2]		M/29 years	Onset	Clonic limb movements	MRI
			2 weeks	Tonic-clonic right arm shaking followed by tonic extension of the right arm and leg	
Matheus [5]		M/50 years	Early	Tonic-clonic movements in all limbs	MRI
Naganuma [6]	Patient 1	M/76 years	Onset	Clonic movements of both arms	MRI
	Patient 2	M/70 years	Onset	Generalized clonic seizure	MRI
Bain [7]		M/2 days	Onset	Right-hand sided clonic movement	MRI
Our case		M/71 years	Onset	Clonic movements of both arms	MRI

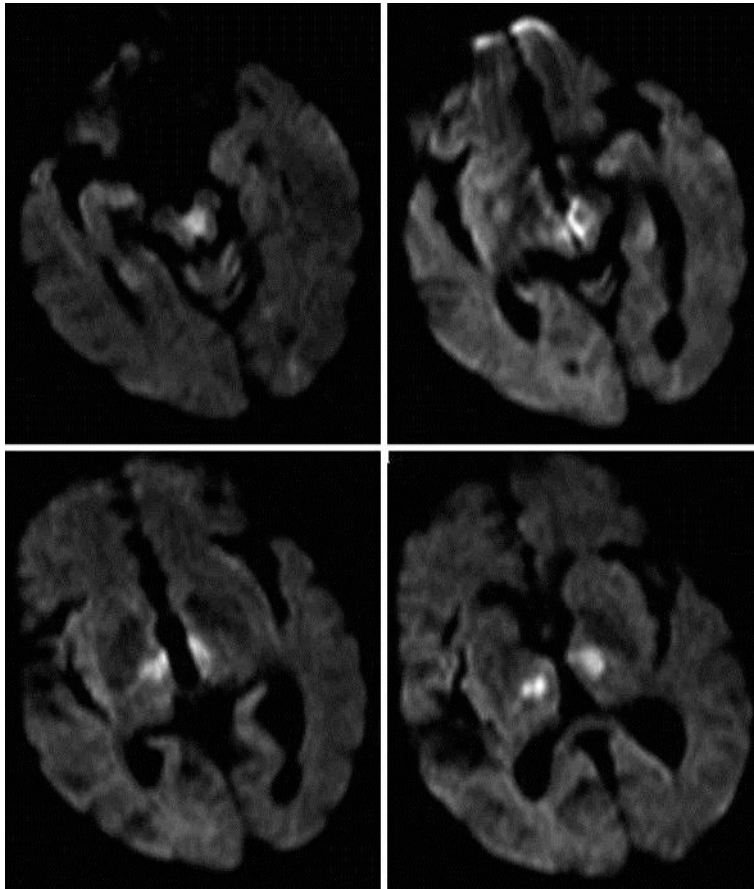


Fig. 1. Diffusion-weighted magnetic resonance imaging demonstrating acute bilateral paramedian thalamic and midbrain infarction.

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